Acid Secretion (μ eq./mg dry wt./hr) (24 experiments)	
Regression equation	$\log H = -(4{\cdot}418 \pm 0{\cdot}576) \cdot 10^3/T + 14{,}037 \pm 0{\cdot}204$
Standard error of estimate of log H	0·85
Correlation coefficient (log H vs. $1/T$)	22,000 ± 2,600 cal./mole
Energy of activation ($-\Delta E$)	3·2 (2·8 – 3·8)
Temperature coefficient (Q_{10})	
Oxygen Consumption (μl/mg dry wt./hr) (25 experiments)	
Regression equation	$\log\lvert Q_{O_2}\rvert = -(3{\cdot}005 \pm 0{\cdot}321) \cdot 10^3/T + 10{\cdot}466 \pm 0{\cdot}113$
Standard error of estimate of $\log\lvert Q_{O_2}\rvert$	0·89
Correlation coefficient ($\log_2\lvert Q_{O_2}\rvert$ vs. $1/T$)	13,700 ± 1,470 cal./mole
Energy of activation ($-\Delta E$)	2·2 (2·0 – 2·4)
Temperature coefficient (Q_{10})	

of these parameters must be judged with caution[1]. The high order of c. 2·5 for Q_{10} means that there is about $10\,\%$ ($=\sqrt[10]{2{\cdot}5}$) increase in secretion rate (and O_2 uptake) per degree temperature increase.

The relation between HCl *production rate* (K^H) *and* O_2 *consumption rate* (K^{O_2}) at any one temperature can be written in logarithmic form (cf.the Arrhenius equation above) as

$$\log \frac{(K^H)}{(K^{O_2})} = (A^H - A^{O_2}) \cdot \frac{1}{T} + (C^H - C^{O_2})$$

where A is an abbreviation for $(-\Delta E/4{\cdot}575)$. As $1/T$ varies very little over the middle range of the temperature interval—the "physiological" one—it may be justified to approximate the right hand member to a constant, B. Hence, one can write $(K^H)/(K^{O_2}) = 10^B$, i.e. there is *approximately linear relation between the acid output and oxygen consumption rate.* The constant 10^B is of the order 0·1 (with a probable variation 0·05–0·2), in other words, *1 µl* O_2*-uptake corresponds to 0·1 µeq.* H^+ *ions (approximately).*

Energy considerations:—It has been calculated (cf. for instance[1]) that the production of 1 liter of gastric juice (being 0·1 normal in HCl) requires at minimum c. 900 cal., i.e. $9{\cdot}10^3$ cal./eq. Furthermore, it is generally assumed that 1 ml O_2-uptake corresponds to 5 cal., hence it is possible to calculate the *thermodynamic efficiency to approximately 20 per cent* (i.e. $0{\cdot}1 \cdot (9{\cdot}10^3 \cdot 10^{-6}/5{\cdot}10^{-3}) = 0{\cdot}18$). This figure is amazingly high especially when considering the fact that the HCl producing cells are only a fraction of all O_2 consuming mucosa cells (cf. CRANE and DAVIES[2] and DAVIES[3] p. 616. TEORELL[4] found for the cat's stomach only 6 to 9 per cent).

Finally it is of interest to express the relation (H^+ *output*) : (O_2 *uptake*) in terms of Q_{HCl} and Q_{O_2} as done by DAVIES and collaborators. As Q_{HCl} at N.T.P. is equal to 22·4 (μ eq. H^+/mg dry wt./hr) one finds for our material of frog gastric mucosa experiments that $Q_{HCl}/\lvert Q_{O_2}\rvert$ *is of the order 2·2* (probable variation 1·1 to 4·4). Although this ratio falls somewhat lower than the majority of those reported by CRANE and DAVIES, it is nevertheless high and being > 1 one may perhaps say that our results seem to confirm DAVIES' statement, that the H^+ ions can not directly originate from oxidative degradation of a substrate known to take part in intermediate metabolism (l. c., p. 617).

The mean Q_{HCl} at 25°C of 3·5 (probable variation 2·2–5·6) is also in good agreement with values published by DAVIES and collaborators.

The mucosa potential has been measured in all cases reported here, it ranged from a few mV to c. 30 mV at maximum (the serosa side positive in the external circuit). Although a covariation between secretion rate and potential could be traced in some single experiments, it was not possible, in our material, to find such a pronounced general relationship between potential and H^+ production, as has recently been argued by REHM[1] and CRANE, DAVIES and LONGMUIR[2].

More complete reports and discussions will be published elsewhere.

TORSTEN TEORELL

Institute of Physiology, University of Uppsala, July 20, 1949.

Zusammenfassung

Die Geschwindigkeit der HCl-Bildung und des O_2-Verbrauchs der isolierten Magenschleimhaut des Frosches sind als Funktionen der Temperatur bestimmt worden. Der Temperaturkoeffizient Q_{10} bewegte sich für die HCl-Bildung in der Größenordnung um 3,2, für den O_2-Verbrauch um 2,2, was einer «Aktivierungsenergie» von ca. 22000 bzw. 14000 Kal./Mol entspricht. Der thermodynamische «Nutzeffekt» der Säuresekretion betrug ≧ 20 v. H. Die Beziehungen zwischen Säurebildung und O_2-Verbrauch werden in bezug auf die Energiequellen und auf die Quellen für die Wasserstoffionenbildung besprochen. Einige Ergebnisse der Potentialmessung an der Schleimhaut werden erwähnt.

[1] See for instance: H. BULL, *Physical Biochemistry*, p. 23, (New York, 1943). A. KANITZ, *Temperatur und Lebensvorgänge* (Berlin 1915). – Y. BELEHRADEK, *Temperature and Living Matter* (Berlin, 1935).

[2] E.E. CRANE and R. E. DAVIES, Proc. Biochem. Soc. (24 Sept. 1948).

[3] R.E. DAVIES, Biochem. J. *42*, 609 (1948).

[4] T. TEORELL, Skand. Arch. Physiol. *66*, 225 (270) (1933).

[1] W.S. REHM, Amer. J. Physiol. *141*, 537 (1944); *144*, 115 (1945).

[2] E.E. CRANE, R.E. DAVIES, and N.M. LONGMUIR, Biochem. J. *43*, 321 (1948).

Effect of ultrasonic vibration on muscle fibres *in vitro*

SZENT-GYÖRGYI[1] and his coworkers[2] have accumulated evidence that the muscle's contractile substance is composed of two protein components, myosin and actin.

[1] A. SZENT-GYÖRGYI, Stud. Inst. Med. Chem. Univ. Szeged. *3*, 76 (1943).

[2] F. B. STRAUB, Stud. Inst. Med. Chem. Univ. Szeged. *2*, 3 (1942).

Myosin can be removed from the fibrils with Weber's solution, actin by treatment with KI solution.

MATOLTSY and GERENDÁS[1] in the course of optical investigation with polarized light of fibres treated with these solvents have demonstrated that, coincidentally with the dissolution of the actomyosin, the fibre's birefringence gradually disappears and a negative double refraction takes its place, as a negative birefringent nucleoprotein (N-protein) remains in the fibres.

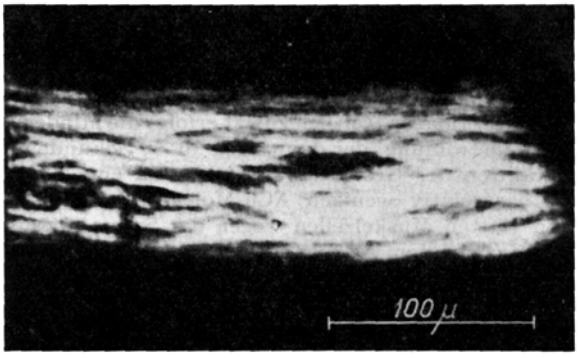

Fig. 1. – Striated muscle fibre irradiated with ultrasonic waves. Longitudinal splitting.

GARAY[2] has shown that ultrasonic irradiation exercizes an extensive depolimerizing effect at the myosin threads. He concludes from this that a similar splitting effect might be brought about on the muscle filaments as well.

In the present experiments we investigated what changes take place in the structure of untreated fibres as an effect of ultrasonic action and what changes we obtain if, before ultrasonic irradiation, the contractile proteins are dissolved as above. In these experiments we irradiated the fibres suspended in physiological NaCl and in $p_H = 7{\cdot}2$ phosphat-buffer for 10 minutes with ultrasonic vibration of 300 kHz frequency and an intensity of 10 watt/cm^2. The ultrasonic waves were produced with a generator constructed by GARAY[3].

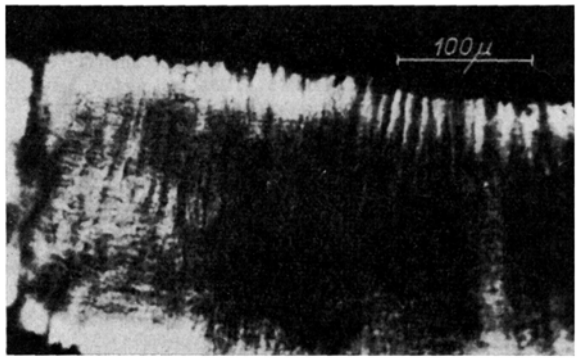

Fig. 2. – The effect of ultrasonic irradiation after the dissolution of contractile proteins. Transversal splitting.

The polarization-microscopic photographs made from the preparations show that the untreated striated muscle fibres under ultrasonic action fray apart longitudinally (Fig. 1)—fibrillarily—while those fibres which in consequence of dissolution no longer contain actomyosin split up perpendicularly to the length of the fibre (Fig. 2). It is characteristic of the splittings that they run parallel and nearly equidistant (5–6 μ).

We explain the phenomenon by the assumption that in the untreated fibres the chemical bond of the polypeptid chain of the contractile protein is stronger than the force holding the fibrile together sideways, and that therefore as an effect of the ultrasonic impulse the weaker, latitudinal bonds break up.

If, however, we break the longitudinally running polypeptid chain by treating it with solvents and dissolve it, the longitudinal coherence of the fibres will be diminished. In this case the fibres split up sideways under irradiation, which proves that the forces of longitudinal cohesion do not break up in consequence of treatment with solvents. Considering that, according to MATOLTSY and GERENDÁS (l. c.), the actomyosin dissolves out of the fibres upon treatment with solvents, but the N-protein remains, it seems possible that it is the N-protein which causes the fibril's diagonal cohesion.

We were unable to observe the phenomenon in heart muscle. Namely, under ultrasonic action these fibres fall apart without any special characteristics. Smooth muscle fibres under ultrasonic action split up sideways even without dissolution of the contractile substance, and a "pseudo striation" can be seen (Fig. 3) with periods of 3–6 μ.

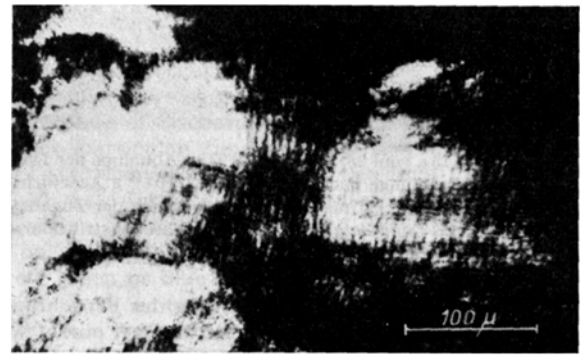

Fig. 3. – Smooth muscle (chicken gizzard) with "pseudo striation" formed as an effect of ultrasonic irradiation.

Protracted (60 minute) ultrasonic action causes complete disorganization in all three types of muscle.

Our investigation will be published in detail elsewhere.

K. GARAY and M. GERENDÁS

Biochemical Institute, University of Budapest, and Hungarian Biological Research Institute, Tihany, Hungary, October 1, 1948.

Zusammenfassung

Die Wirkung von Ultraschall auf isoliertes Muskelgewebe wird untersucht und dabei folgendes gefunden:

Im quergestreiften Muskel, in dem das Actomyosin nicht in der üblichen Weise entfernt worden ist, spalten sich die Muskelfibrillen in der Längsachse. Nach Entfernung des Actomyosins gibt es Aufspaltungen senkrecht zur langen Achse der Fibrillen. Die entstehenden parallelen Spalte liegen in Abständen von 4–8 μ.

Im glatten Muskel gibt es, in Abständen von 3–4 μ, senkrecht zu den Fibrillen verlaufende Spalte, auch ohne daß das Actomyosin entfernt worden ist.

Der Herzmuskel wird durch Ultraschall nicht nennenswert beeinflußt.

Die Bedeutung der Befunde wird diskutiert.

[1] A. G. MATOLTSY and M. GERENDÁS, Nature *159*, 502 (1947); Hung. acta physiol. *1*, 116 (1948); Hung. acta physiol. *1*, 124 (1948); Hung. acta physiol. *1*, 128 (1948).

[2] K. GARAY, Hung. acta physiol. (in the press).

[3] K. GARAY and L. BERTA, M. Orvosi Arch. 4–5 sz 44 (1943).